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Demodicidosis on the arms of a patient with pemphigus foliaceus

To the Editor: Demodex mites are common commensals of the human pilosebaceous follicle that use sebum as nourishment.[1] They are most frequently found on the face, where the number of sebaceous glands is greatest, but they have also been observed on the ear canal, scalp, neck, back, chest, nipples, buttocks, and penis.[2] To our knowledge, there is only 1 case report of demodectic mites on the extremities (legs).[2] Here we report a case of demodicidosis on the arms.

A 56-year-old Korean man presented with a several-day history of multiple pustules localized on both arms. The lesions were asymptomatic. Pemphigus foliaceus was diagnosed by skin biopsy 2 years earlier and he had been undergoing various immunosuppressive treatments such as mycophenolate mofetil (MMF), oral corticosteroid, antihistamine, topical corticosteroids, and topical calcineurin inhibitors in the 2 years since the diagnosis. At the time of the initial appearance of the arm lesions, the patient was on 360 mg of MMF and 6 mg of methylprednisolone intermittently for 2 months. The lesions seemed to increase in number with oral intake of MMF and decrease with discontinuation.

Physical examination revealed numerous discrete 2- to 3-mm erythematous follicular papules and pustules on both antecubital fossae (Fig 1). A potassium hydroxide preparation of scrapings from the erythematous pustules and scales showed the presence of many *Demodex* mites (Fig 2). We diagnosed this case as a *Demodex* infestation in a patient with pemphigus foliaceus. Treatment was successful, albeit slow, over 3 months, with twice-daily applications of topical metronidazole and benzene hexachloride to the affected areas. We could not use ivermectin because it is not available in Korea.

Demodex mites are considered normal skin fauna, and there is no consensus to what degree the mites are causative of skin pathology and how they might contribute to disease.[1] In general, *Demodex* mites are thought to play a pathogenic role when present in large numbers or with penetration into the dermis.[3] In specific environments such as immunologic deficiency, they can proliferate and provoke cutaneous lesions.[4] The patient had previously taken a variety of immunosuppressive drugs for pemphigus foliaceus treatment, which could be an important predisposing factor for an increase in the number of *Demodex* mites on unusual sites. One of the multiple effects of MMF on the immune system is an inhibition of T-lymphocyte proliferation,[5] which can lead to mite invasion and development of clinical symptoms.[4] Demodicidosis can present in the extremities. Therefore, demodicidosis should be included in the differential diagnosis of erythematous or pustular follicular eruptions of the extremities, particularly in patients with immunodeficiency.

Fig 1. Demodex infestation in a patient with pemphigus foliaceus.

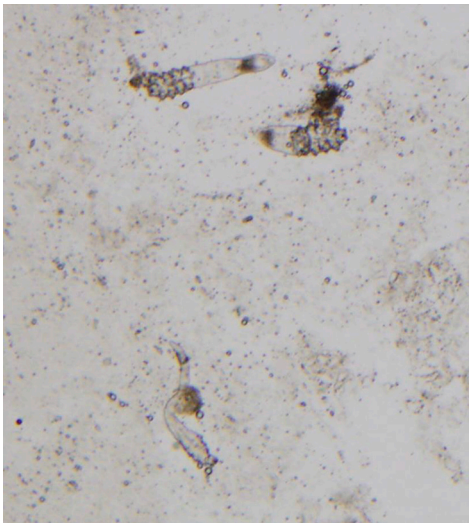

Fig 2. Potassium hydroxide scraping from *Demodex* folliculitis of antecubital fossa. (Original magnification: ×50.)

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Primary cutaneous nocardiosis in an immunocompetent host following laser resurfacing

To the Editor: A 66-year-old immunocompetent woman presented with a 3-month history of a nonhealing left infraorbital ulcer. The lesion began as a pustule following erbium:YAG laser resurfacing performed to improve cosmesis after an upper and lower blepharoplasty. Over several weeks the lesion evolved into a shallow, nonhealing ulceration. Initially, the patient was treated with minocycline, cephalexin, and topical mupirocin without improvement. On presentation to our dermatology clinic, the patient was being treated with oral prednisone 60 mg daily, topical desoximetasone 0.25% ointment, and valacyclovir 1000 mg twice daily.

On physical examination, a 1.4-cm oval shallow ulceration without purulence or exudate was noted on the left infraorbital rim (Fig 1). Skin biopsy revealed ulceration with a mixed inflammatory infiltrate. Gram stain demonstrated gram-positive, focally branching, bacilli (Fig 2). Periodic acid—Schiff and acid-fast bacilli stains were negative; however, mycobacterium tuberculosis polyclonal antibody stain (Biocare Medical PP140AA) showed dot-like positivity. No evidence of herpes viral change was noted.

On follow-up, prednisone was gradually discontinued and minocycline was started for presumed atypical mycobacterial infection. Another biopsy was obtained for bacterial, fungal, and mycobacterial culture. The receiving microbiology lab was notified in advance of the possibility of atypical mycobacteria and *Nocardia* species. Bacterial cultures were initially reported as normal skin flora. The microbiology laboratory was asked to specifically look for *Nocardia*. Several days later, *Nocardia asteroides* complex was isolated. Fungal and mycobacterial cultures were negative at 6 weeks. Trimethoprim-sulfamethoxazole DS 3 times daily was initiated and the patient was referred to an infectious disease physician for a workup to identify potential sources of infection. The workup included magnetic resonance imaging of the brain, as well as computed tomography examination of the chest; no systemic source of infection was identified. Marked improvement was noted at 2 weeks (Fig 2), although a mild ectropion developed at 3 months.

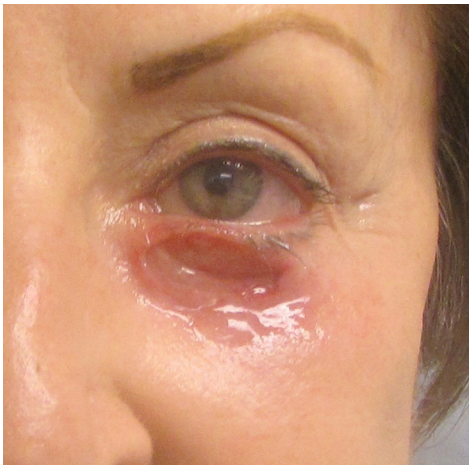

Fig 1. Primary cutaneous nocardiosis left infraorbital ulceration.

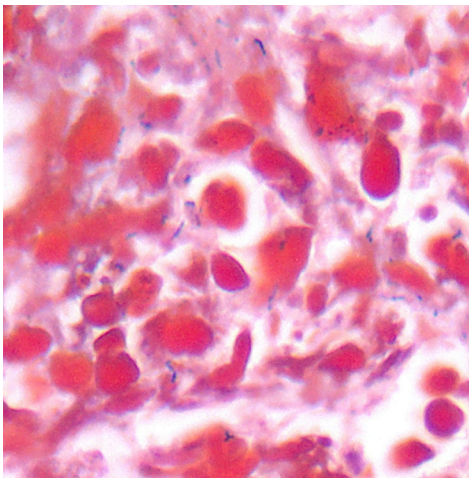

Fig 2. Primary cutaneous nocardiosis Gram stain demonstrating gram-positive, focally branching bacilli.